A/65/46

United Nations

# Report of the United Nations Scientific Committee on the Effects of Atomic Radiation

## Fifty-seventh session
## (16-20 August 2010)

**General Assembly**
**Official Records**
**Sixty-fifth session**
**Supplement No. 46**

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United Nations • New York, 2010

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ISSN 0255-1373

[15 September 2010]

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# Chapter I

## Introduction

1. Exposure to ionizing radiation arises from sources such as medical diagnostic and therapeutic procedures; nuclear weapons testing; radon and other natural background radiation; nuclear electricity generation; accidents such as the one at Chernobyl in 1986; and occupations that increase exposure to artificial or natural sources of radiation.

2. Since the establishment of the United Nations Scientific Committee on the Effects of Atomic Radiation by General Assembly resolution 913 (X) of 3 December 1955, the mandate of the Committee has been to undertake broad assessments of the sources of ionizing radiation and its effects on human health and the environment.[1] In pursuit of its mandate, the Committee thoroughly reviews and evaluates global and regional exposures to radiation, and also evaluates evidence of radiation-induced health effects in exposed groups, including survivors of the atomic bombings in Japan. The Committee also reviews advances in the understanding of the biological mechanisms by which radiation-induced effects on health or on the environment can occur. Those assessments provide the scientific foundation used, inter alia, by the relevant agencies of the United Nations system in formulating international standards for the protection of the general public and workers against ionizing radiation;[2] those standards, in turn, are linked to important legal and regulatory instruments.

[1] The United Nations Scientific Committee on the Effects of Atomic Radiation was established by the General Assembly at its tenth session, in 1955. Its terms of reference are set out in resolution 913 (X) of 3 December 1955. The Committee was originally composed of the following Member States: Argentina, Australia, Belgium, Brazil, Canada, Czechoslovakia, Egypt, France, India, Japan, Mexico, Sweden, Union of Soviet Socialist Republics, United Kingdom of Great Britain and Northern Ireland and United States of America. The membership of the Committee was subsequently enlarged by the Assembly in its resolution 3154 C (XXVIII) of 14 December 1973 to include the Federal Republic of Germany, Indonesia, Peru, Poland and the Sudan. By its resolution 41/62 B of 3 December 1986, the Assembly increased the membership of the Committee to a maximum of 21 members and invited China to become a member.

[2] For example, the international basic safety standards for protection against ionizing radiation and for the safety of radiation sources, currently co-sponsored by the International Labour Organization, the Food and Agriculture Organization of the United Nations, the World Health Organization (WHO), the International Atomic Energy Agency (IAEA), the Nuclear Energy Agency of the Organization for Economic Cooperation and Development and the Pan American Health Organization.

# Chapter II

## Deliberations of the United Nations Scientific Committee on the Effects of Atomic Radiation at its fifty-seventh session

3. The Committee held its fifty-seventh session in Vienna from 16 to 20 August 2010.[3] Norman Gentner (Canada), Wolfgang Weiss (Germany) and Mohamed A. Gomaa (Egypt) served as Chair, Vice-Chair and Rapporteur, respectively. The session had been twice unavoidably postponed, once owing to an unforeseen personal crisis for the Secretary of the Committee and subsequently because of worldwide flight disruptions following the April 2010 volcanic eruptions in Iceland. The Committee acknowledged and supported the two letters of the Chair of the Committee to the President of the General Assembly informing him of the reasons for the postponements.[4]

4. With regard to the report with scientific annexes that it had approved in 2006,[5] the Committee expressed its dissatisfaction that volume II, containing the scientific annexes entitled "Non-targeted and delayed effects of exposure to ionizing radiation", "Effects of ionizing radiation on the immune system" and "Sources-to-effects assessment for radon in homes and workplaces", had not been published until July 2009. Moreover, with regard to the report with scientific annexes that it had approved in 2008,[6] it also expressed its dissatisfaction that volume I, with scientific annexes entitled "Medical radiation exposures" and "Exposures of the public and workers from various sources of radiation" had not been published until July 2010 and that volume II, with scientific annexes entitled "Radiation exposures in accidents", "Health effects due to radiation from the Chernobyl accident" and "Effects of ionizing radiation on non-human biota", was still not published. The Committee regards the late publication as intolerable, because Member States and relevant organizations[7] rely on the information contained in those reports, to which the Committee members had contributed invaluable expertise, and because the technical basis of the reports would be outdated when they appeared. The Committee emphasized to the secretariat that it was especially important that the new material on the Chernobyl accident be published well before the twenty-fifth anniversary of the accident.

5. The secretariat reported that the delays were traceable in part to inadequate staffing and to a lack of sufficient, assured and predictable funding, and to

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[3] The fifty-seventh session of the Committee was also attended by observers for Belarus, Finland, Pakistan, the Republic of Korea, Spain and Ukraine, in accordance with General Assembly resolution 64/85, para. 14, and observers for the United Nations Environment Programme, WHO, IAEA, the European Commission, the International Commission on Radiological Protection and the International Commission on Radiation Units and Measurements.

[4] A/64/223 and A/64/796.

[5] See *Official Records of the General Assembly, Sixty-first Session, Supplement No. 46* (A/61/46).

[6] See *Official Records of the General Assembly, Sixty-third Session, Supplement No. 46* (A/63/46).

[7] For example, at its fifty-third regular session, the IAEA General Conference, in its resolution GC(53)/RES/10, entitled "Measures to strengthen international cooperation in nuclear, radiation, transport and waste safety", encouraged the IAEA secretariat to continue to take account of the scientific information provided by the United Nations Scientific Committee on the Effects of Atomic Radiation when developing Agency safety standards.

publishing procedures within the United Nations. The Committee suggests that the General Assembly might request the United Nations Secretariat to streamline the procedures for publishing the Committee's reports as sales publications, recognizing that, while maintaining quality, the timeliness of their publication is paramount to meet expected accomplishments approved in the programme budget.

6. The Committee noted that the General Assembly, in its resolution 63/89, had requested the Secretary-General, in formulating his proposed programme budget for the biennium 2010-2011, to consider all options, including the possibility of internal reallocation, to provide the Scientific Committee with the resources outlined in paragraphs 48 and 50 of the report of the Secretary-General addressing the financial and administrative implications of increased Committee membership, staffing of the professional secretariat and methods to ensure sufficient, assured and predictable funding.[8] The Committee noted with satisfaction that an additional Professional post, at the P-4 level, was included in the programme budget for the biennium 2010-2011. This is in line with the recommendations contained in the report of the Secretary-General, and will finally address the concern that reliance on a single post at the Professional level in the Committee's secretariat had left the Committee seriously vulnerable and had hampered the efficient conduct of its approved programme of work.

7. The Committee developed and approved for submission to the General Assembly a scientific report that summarizes low-dose radiation effects on health, including a synthesis of the Committee's detailed findings on the mechanisms of radiation actions at low doses (chap. III below). The Committee considers that there would be merit in disseminating this summary widely in all official languages of the United Nations.

8. The Committee reviewed substantive documents on the attributability of health effects to radiation exposure and on uncertainty in radiation risk estimation. With regard to the attributability of health effects at low doses, the Committee decided that there was a need to consider the ability to attribute scientifically risk and effects at both high and low doses, and that the ability to attribute should be clarified both for populations and for individuals. A definitive document on this issue will be taken up at the fifty-eighth session of the Committee.

9. The Committee also reviewed preliminary documents on the assessment of levels of radiation from electrical energy production, and its methodology for estimating exposures due to discharges. The Committee recognized that its assessments of radiation exposures from electricity generation, while up to date and detailed for the nuclear fuel cycle, were out of date for the enhanced levels of naturally occurring radioactive material associated with the use of fossil fuels, and moreover had never been assessed in a comparable way for renewable energy sources. The methodology would need to be updated in order to conduct the assessment in a fair manner, and a literature review and data collection were initiated.

10. The Committee approved a strategy developed to improve data collection, analysis and dissemination. This strategy is based on the development of electronic solutions, targets specific countries and entails close collaboration with other

[8] A/63/478 and Corr.1.

networks, in particular those of the International Atomic Energy Agency (IAEA) and the World Health Organization (WHO). The Committee suggests that the General Assembly might (a) encourage Member States, the organizations of the United Nations system and other relevant organizations concerned to provide further relevant data about doses, effects and risks from various sources of radiation, which would greatly help in the preparation of future reports of the Committee to the General Assembly; and (b) encourage IAEA, WHO and other relevant organizations to collaborate with the Committee secretariat to establish and coordinate the arrangements for periodic collection and exchange of data on radiation exposures of the general public, workers and, in particular, medical patients.

11. The Committee also reviewed plans for conducting work on medical radiation exposures, biological effects of selected internal emitters, enhanced exposures to natural sources of radiation due to human activities, development of a knowledge base on radiation levels and effects, and improving public information. It decided that the work on selected internal emitters should focus on tritium and uranium; that the focus of assessments of enhanced exposures to natural radiation sources ought to change to avoid overlaps with other work; and that the development of a knowledge base on radiation levels and effects ought to follow after enhancing collection of exposure data.

12. In addition to reviewing progress on the existing programme of work, the Committee considered proposals for its future programme of work. It decided to conduct some preparatory investigations into the merits and appropriateness of preparing substantive assessments of radiation effects and risks specifically for children, and of the epidemiology of exposures of the public to natural and artificial environmental sources at low doses and low dose rates.

13. The Committee recognizes that in order to accelerate the conduct of its programme of work, voluntary contributions to the general trust fund established by the Executive Director of the United Nations Environment Programme to receive and manage voluntary contributions to support the work of the Committee would be beneficial. The Committee suggests that the General Assembly might encourage Member States to consider making voluntary contributions to the general trust fund for these purposes.

14. The Committee discussed in detail the reminder in paragraph 13 of resolution 64/85 to

> continue its reflection on how its current, as well as its potentially revised, membership could best support its essential work, including by developing, with the participation of the observer countries, detailed, objective and transparent criteria and indicators to be applied equitably to present and future members alike, and to report its conclusions.

The Committee's conclusions on this matter are presented in addendum 1 to the present report.

15. The Committee decided to hold its fifty-eighth session in Vienna from 23 to 27 May 2011. New officers were elected to guide the Committee at its fifty-eighth and fifty-ninth sessions: Wolfgang Weiss (Germany), Chair; Carl-Magnus Larsson (Australia), Vice-Chair; and Mohamed A. Gomaa (Egypt), Rapporteur.

# Chapter III

## Scientific report: summary of low-dose radiation effects on health

16. In its scientific work, the Committee pays particular attention to reviewing information on the health effects of exposure to ionizing radiation. A key objective is to provide evidence-based estimates of the risks to human health from exposure to low doses and low dose rates of radiation that may be received, throughout the world, by the general public, workers and patients undergoing medical procedures. For these purposes, the Committee defines low doses as those of 200 milligrays (mGy) or less and low dose rates as 0.1 mGy per minute (averaged over one hour or less) for radiations such as external X-rays and gamma rays;[9] the Committee notes, however, that different values are used to define low dose and low dose rate for other purposes. The present report consolidates and summarizes, in simple terms, the Committee's detailed understanding of these key areas up to 2006 as presented in its reports to the General Assembly.[10, 11, 12]

17. The Committee has produced radiation risk estimates for cancer and hereditary effects. More recently, there is increasing evidence of low-dose radiation exposure leading to increased incidence of cataracts. There is also some concern raised by findings of elevated incidence of circulatory disease in populations irradiated at high doses. These non-cancer diseases arise naturally and can be relatively common in the general population. Many difficulties are encountered in attributing specific cases of disease to low-dose radiation exposure including:

(a) The lack of specificity in the type or characteristics of disease induced by radiation exposure;

(b) The long delay (years or decades) between exposure and disease presentation;

(c) The high spontaneous incidence of diseases associated with radiation in the ageing general population.

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[9] When ionizing radiation passes through matter, including living tissue, it deposits energy that ultimately produces ionization and excitation in the matter. The amount of energy deposited divided by the mass of tissue exposed is called the absorbed dose and is usually measured in units known as milligrays (mGy).

[10] *Effects of Ionizing Radiation: United Nations Scientific Committee on the Effects of Atomic Radiation — 2006 Report to the General Assembly, with Scientific Annexes A and B*, vol. I (United Nations publication, Sales No. E.08.IX.6 and corrigendum).

[11] *Effects of Ionizing Radiation: United Nations Scientific Committee on the Effects of Atomic Radiation — 2006 Report to the General Assembly, with Scientific Annexes C, D and E*, vol. II (United Nations publication, Sales No. E.09.IX.5).

[12] *Hereditary Effects of Radiation: United Nations Scientific Committee on the Effects of Atomic Radiation — 2001 Report to the General Assembly, with Scientific Annex* (United Nations publication, Sales No. E.01.IX.2).

Responding to a specific request of the General Assembly,[13] the Committee is preparing a comprehensive report addressing the ability to attribute scientifically health effects to low-dose radiation exposure.

18. The information available to the Committee falls into two general classes:

(a) The results of studies of excess disease in populations irradiated generally at moderate or high doses (i.e. obtained through epidemiological studies);

(b) The results of studies using experimental models of disease (i.e. with animals and cultured cells, often coupled with studies of radiation effects at the subcellular, biochemical and molecular levels) that can provide information on the mechanism through which the biological effect or disease arises.

19. Because the diseases of interest can be relatively common and their incidence may be influenced by factors other than radiation exposure, epidemiological observations are frequently unable to reveal clear evidence of radiation-associated increased incidence at low doses. For this reason, the Committee has sought to make full use of recent advances in knowledge from experimental studies and in the understanding of the mechanistic basis for human disease. In developing health risk estimates, the Committee considers populations, not individuals. Increasing evidence suggests that genetic and other factors can affect risk of disease; such factors may be better understood in the future.

## A. Radiation-induced cancer

20. Cancer is a general term used to describe major disturbances in the growth pattern of primitive cells in body organs. These primitive cells normally develop and divide in a coordinated way to form the specialist cells of the organ, but abnormal growth and arrested development can lead to a mass of cells in a given organ, which is termed a solid tumour. Such abnormal growth or development in primitive bone marrow and lymphatic cells can lead, respectively, to leukaemia and lymphoma. Depending upon the organ in question, unchecked tumour growth and further cellular changes can lead to the spread of malignant disease, which is frequently fatal. There is strong epidemiological evidence that exposure of humans to radiation at moderate and high levels can lead to excess incidence of solid tumours in many body organs and of leukaemia. There is also growing information on the cellular/molecular mechanisms through which these cancers can arise.

21. Cancers are due to many causes, are frequently severe in humans, and are common, accounting for about one quarter of deaths in developed countries and a growing burden of deaths in developing countries. As will be seen below, any increase in cancer incidence thought to be caused by low-dose radiation exposures is modest by comparison.

### 1. Epidemiological studies

22. The Committee has used for a number of years a system of rolling reviews of all the studies of radiation-associated cancer incidence in irradiated human populations. Particular attention has been given to the soundness of study design,

[13] Resolution 62/100, para. 6.

including consideration of a wide range of potential confounding factors and of the statistical power of any such study to reveal excess radiation-associated cancer incidence. The Committee's analysis includes an assessment of statistical power, the potential for systematic error and other sources of uncertainty, including those associated with the radiation doses received. The Committee also considers the most appropriate method for estimating risk in populations with characteristics differing from those of the studied populations (e.g. the method for estimating the risk to an irradiated United States population based on the estimates derived from the survivors of the atomic bombings in Japan).

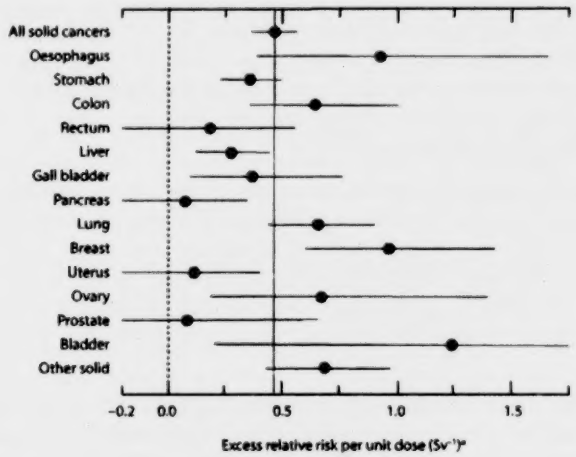
23. Epidemiological information on radiation-associated cancer incidence comes from studies of the survivors of the atomic bombings in Japan, of groups exposed in their working environment, of patients irradiated in the course of medical procedures and of people exposed to environmental sources of radiation. In recent years, it has also been possible to detect excess lung cancer incidence in groups of people exposed in their homes to the naturally occurring radioactive gas radon and its progeny.

24. In reviewing all these studies, the Committee has judged that the single most informative set of data on whole-body radiation exposure comes from studies of the survivors of the atomic bombings in Japan in 1945. The atomic-bombing exposures were predominantly high-dose-rate gamma radiation, with a small contribution from neutrons. The Committee has used these data to assess the risks of radiation-associated solid cancer together with the risk of leukaemia and of lymphoma. Although statistical and other uncertainties limit the analyses of all data sets, it has also been possible to examine trends in radiation risk associated with sex, age at exposure and time since exposure and also how risks may differ for populations from different parts of the world. There are some cancer types for which there is no evidence of an excess risk after radiation exposure and others for which an excess is seen only after high doses of radiation. Figure I shows the variation in sensitivity to the induction of solid cancers arising at 13 different sites in the body, based on mortality among the survivors of the atomic bombings in Japan. The figure clearly shows substantial differences in the risk of cancer induction for various organs.[14]

[14] As of December 2000, 45 per cent of the cohort of 86,611 survivors were still alive. Of 10,127 deaths from solid cancer due to all causes, 479 would be estimated to be associated with the radiation exposure from the bomb detonations, as would 93 leukaemia deaths out of 296 leukaemia deaths from all causes.

Figure I

**Estimates of solid cancer mortality risk for different organs from studies of the survivors of the atomic bombings in Japan**




*Source*: *Effects of Ionizing Radiation: United Nations Scientific Committee on the Effects of Atomic Radiation — 2006 Report to the General Assembly, with Scientific Annexes A and B*, vol. I (United Nations publication, Sales No. E.08.IX.6 and corrigendum), annex A, fig. XI.

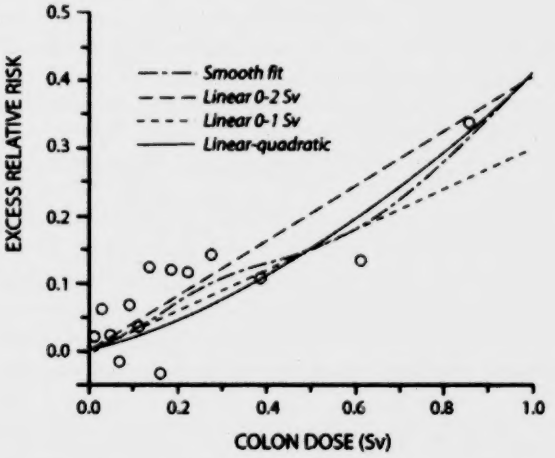
[a] While grays (Gy) are used to express absorbed dose, in estimating biological effect a weighted quantity is used, which takes account of the fact that different kinds of radiation have different biological effects for the same amount of energy deposited. The weighted quantity is expressed here in sieverts (Sv). The horizontal bars represent 90 per cent confidence intervals.

25. The Committee has used the epidemiological data to examine the relationship between the radiation dose received and the risk of cancer induction, i.e. the dose-response relationship. Excess relative risk is a measure of the size of the increase in cancer risk in the study population due to the radiation at given doses (larger numbers indicate higher risk). The data from the survivors of the atomic bombings in Japan for all solid cancers combined provides the clearest picture of this relationship; this is shown in figure II. The dose-response relationship for mortality at low doses shown in figure II may be described by both a linear and a curvilinear function. Statistically significant elevations in risk are observed at doses of 100 to 200 mGy and above. Epidemiological studies alone are unlikely to be able to identify significant elevations in risk much below these levels. It is a complex process to extract from all informative studies an overall estimate of the lifetime risk of cancer induction from radiation exposure. The Committee has used mathematically based models together with data on the underlying cancer rates in five populations from different regions of the world to address this question, but fully recognizes the uncertainties in these estimates. The Committee's current estimates for the risks of radiation-induced fatal cancers are shown in table 1; these cancer risk estimates are similar to those previously made by the Committee and other bodies. Risk estimates vary with age, with younger people generally being more sensitive; studies of in utero radiation exposures show that the foetus

is particularly sensitive, with elevated risk being detected at doses of 10 mGy and above.

Figure II

**Dose response for solid cancer mortality based on the 2002 studies of the survivors of the atomic bombings in Japan**




*Source: Effects of Ionizing Radiation: United Nations Scientific Committee on the Effects of Atomic Radiation — 2006 Report to the General Assembly, with Scientific Annexes A and B*, vol. I (United Nations publication, Sales No. E.08.IX.6 and corrigendum), annex A, fig. IX.

26. The radiation exposures of the survivors of the atomic bombings in Japan were very unlike those of most of the groups of people exposed either to radiation during the course of their work or to environmental sources of radiation. The atomic-bombing survivors were exposed to external radiation from gamma rays and neutrons, generally at high doses over short periods. By contrast, many of the other groups were exposed over long periods to low doses; and sometimes the exposure was from internally incorporated radionuclides. Valuable information on the long-term low-dose exposures to internally incorporated radionuclides has been provided by epidemiological studies of the health of workers at the Mayak nuclear complex in the southern Urals of the Russian Federation, and of the population near the Techa River whose exposure was due to radioactive discharges from that facility. Follow-up of those exposed as a consequence of the Chernobyl accident has provided useful information on the effects of low-dose external radiation exposure, and on the effects of thyroid exposure to radioiodine. Overall, the cancer risk estimates from these studies do not differ significantly from those obtained from the studies of the atomic-bombing survivors in Japan. By contrast, studies on human populations living in areas with elevated natural background radiation in China and India do not indicate that radiation at such levels increases the risk of cancer. New data continue to emerge from these and other studies. The Committee will continue to keep these under review.

Table 1
**Excess lifetime risk of mortality (averaged over both sexes)**[a]

| *Acute dose (Gy)* | *Solid cancers combined (percentage at specified dose)* | *Leukaemia (percentage at specified dose)* |
|---|---|---|
| 0.1 | 0.36-0.77 | 0.03-0.05 |
| 1.0 | 4.3-7.2 | 0.6-1.0 |

*Source: Effects of Ionizing Radiation: United Nations Scientific Committee on the Effects of Atomic Radiation — 2006 Report to the General Assembly, with Scientific Annexes A and B*, vol. I (United Nations publication, Sales No. E.08.IX.6 and corrigendum), annex A, para. 593.

[a] An excess lifetime risk of 1.0 per cent equates to 1 additional case per 100 people.

## 2. Mechanistic studies

27. An understanding of the mechanisms of cancer development after radiation exposure can assist in the interpretation of epidemiological data, particularly in projecting downward to estimate risks for low doses and low dose rates. Since 2000, the Committee has placed increased emphasis on reviewing developments in this area.

28. Over many years, studies of the development of cancer have accumulated evidence that, in general, the process starts by the change (mutation) of one or more genes of the DNA of a single "stem-like" cell in a body organ. Subsequent cancer development and the onset of malignancy are believed to proceed in a multistep fashion, and these steps have also been associated with mutation or other changes involving cellular genes.

29. The Committee has reviewed the findings of those studies, together with those of many studies of the cellular and subcellular effects of radiation exposure. The current understanding is that the energy deposited in the cell after irradiation can damage all subcellular components. The main subcellular targets for radiation-associated cellular change are the DNA molecules residing in the chromosomes. The DNA codes for some 25,000 genes that coordinate all functions in each cell, and unless the radiation damage affecting a gene (or group of genes) is repaired correctly, the cell may die. Alternatively, the cell may survive but with DNA mutations that affect cellular behaviour. A small fraction of such mutations can contribute to cancer development. Cells have a number of DNA repair systems that can correct many forms of DNA damage that have been induced spontaneously or by external agents. In a broad sense, the DNA repair systems serve to restore the genetic integrity of a cell. Importantly, the key mutational events in cancer development are frequently dependent on the organ in which the irradiated cell is located and fall into two general categories — small specific mutations in single genes and mutations involving loss of DNA (sometimes spanning more than one gene).

30. Highly sophisticated studies on the ways in which radiation causes damage to cellular DNA, on the cellular systems that act to recognize and repair that damage and on the development of radiation-associated DNA mutations have cast new light on possible mechanisms for cancer development. Radiation can simultaneously

damage both strands of the DNA double helix, often resulting in breakage of the DNA molecule with associated complex chemical changes.

31. This type of complex DNA damage is difficult to repair correctly, and even at low doses of radiation it is likely that there is a very small but non-zero chance of the production of DNA mutations that increase the risk of cancer developing. Thus, the current balance of available evidence tends to favour a non-threshold response for the mutational component of radiation-associated cancer induction at low doses and low dose rates. Information on the nature of radiation-associated mutations suggests that DNA-loss events (gene deletions) will tend to dominate this mutational component. There is also some evidence that the reduction in cancer risk for a given exposure at low doses and low dose rates as compared with that at high doses and high dose rates is associated, at least in part, with cellular capacities in dealing with DNA damage after radiation exposure. An adjustment factor known as the dose and dose-rate effectiveness factor is often used to take into account the comparative reduction in effect due to low doses and dose rates; however, in the 2006 report of the Committee[10] a linear-quadratic model was used directly for extrapolation to estimate risks at low doses, and so no dose and dose-rate effectiveness factor was applicable.

32. The induction and development of cancer after radiation exposure is not simply a matter of the stepwise accumulation of mutations in the DNA of the relevant cells. There have been studies relating to the following hypotheses: (a) that adaptation of cells and tissues to low doses of radiation might cause them to become more resistant to cancer development (adaptive response); (b) that the effects of radiation on the immune systems, which recognize and destroy abnormal cells, could influence the likelihood of cancer development; and (c) that radiation can produce changes that create long-lasting and transmissible effects on the stability of cellular DNA (genomic instability) and/or trigger the transfer of signals from damaged cells to their undamaged neighbours (bystander effects); both genomic instability and bystander effects have been suggested as possible factors that modify radiation-associated cancer risk. These and other modulating factors, such as the induction of inflammatory reactions, could serve to increase or decrease the cancer risk due to radiation exposure.

33. The Committee has reviewed these studies and judges that these processes do not contribute significantly to the interpretation of the epidemiological data.

34. Cancer risk is affected by the nature of the radiation exposure. Radiation of different qualities (e.g. X-rays, beta radiation, alpha particles) differ in their effectiveness at inducing cancer. Furthermore, radiation exposure may be internal, from ingestion or inhalation of radioactive material, or external, from a radiation source such as a diagnostic X-ray examination. The dispersal of radioactive material in the body as a consequence of an intake is complex, and elaborate models are needed to estimate the doses delivered to tissues and their impact on health. Internal exposures were a major component of the overall radiation exposures following the Chernobyl accident. The Committee has specifically addressed the consequences of this event.[15] Risk estimates for internal exposures are also available from epidemiological studies of the workforce at the Mayak nuclear complex in Russia and of several other groups of exposed people.

[15] See *Official Records of the General Assembly, Sixty-third Session, Supplement No. 46* (A/63/46).

35. Finally, the Committee has reviewed the development in knowledge of inherited conditions that lead to increased susceptibility to incurring certain cancers over the normal incidence in individuals. There is some epidemiological evidence from studies of radiotherapy patients that individuals with certain types of these predisposing conditions are at increased risk of incurring cancer following a radiation exposure. The results of experimental studies with cells and animals strengthen this conclusion and suggest that this increased sensitivity to radiation exposure in people who are predisposed to cancer may be more general. Other individual factors (e.g. age, hormone status, immune status) and environmental factors (e.g. exposure to toxins, diet) could contribute to individual radiosensitivity. However, at present this preliminary conclusion is limited to inherited conditions, where excess cancer incidence is clearly apparent in families. These conditions are too rare in populations to influence the Committee's estimates of cancer risk; nevertheless, a lower degree of heritable sensitivity to radiation-associated cancer may be more common.

## B. Heritable effects of radiation exposure

36. Unlike the studies on radiation-associated cancer, epidemiological studies have not provided clear evidence of excess heritable effects of radiation exposure in humans. The largest and most extensive study of this type was conducted using data on children of the survivors of the atomic bombings in Japan. No increase in the frequency of heritable effects was observed in that or any other human study. Therefore, these studies do not allow a direct estimate of any heritable risks of radiation exposure. Neither do they confirm that there is no risk of heritable effects, because detecting a small excess incidence associated with radiation exposure above a fairly high incidence in unirradiated populations (table 2) is difficult. However, the results of those studies are helpful in that they can give an upper boundary for the estimate of any associated risk.

Table 2
**Estimates of the risk of heritable diseases in the next generation due to low-dose exposure of the parent population**

| *Disease class* | *Baseline frequency (per million people)* | *Risk in first generation per unit low-LET [a] dose (per million people exposed to 1 Gy)* |
|---|---|---|
| Dominant (including X-linked diseases) | 16 500 | ~750-1 500 |
| Chromosomal | 4 000 | [b] |
| Chronic multifactorial diseases | 650 000 | ~250-1 200 |
| Congenital abnormalities | 60 000 | ~2 000 |

*Source*: *Hereditary Effects of Radiation: United Nations Scientific Committee on the Effects of Atomic Radiation — 2001 Report to the General Assembly, with Scientific Annex* (United Nations publication, Sales No. E.01.IX.2), annex, table 46.

[a] Types of radiation with a low linear energy transfer (low-LET) include X-rays, gamma rays and beta particles.

[b] Assumed to be subsumed partly under the risk of autosomal dominant and X-linked diseases and partly under that of congenital abnormalities.

37. Whereas the cancer-related effects of irradiation arise in the organs of people directly exposed, heritable effects originate from damage to the DNA of germ cells (sperm and eggs) in the reproductive organs (testes in males and ovaries in females) of these exposed individuals. If such DNA damage creates germ-cell mutations, then these can be passed to the offspring of the irradiated person and onward through future generations. A proportion of these mutations will directly give rise to dominantly inherited diseases. Other mutations operate indirectly by interaction with other genes and lifestyle or environmental factors to produce chronic multifactorial diseases. Both classes of disease arise naturally in any case, contributing to birth defects (congenital abnormalities) in children. Studies of the incidence of congenital malformations involving a large number of newborn children in the areas of high natural background radiation in India and China do not indicate an increase in the frequency of malformations.

38. The clearest demonstrations of the heritable effects of radiation exposure come from extensive experimental studies on animals at high doses, particularly laboratory mice. The Committee has reviewed and analysed the data on mutations from these studies, especially in the light of new information on the types of germ-cell DNA mutations involved (largely DNA losses) and how they relate to heritable effects in the offspring. The Committee has adopted the so-called mutational doubling dose as a measure of the heritable effects of radiation exposure. The doubling dose of radiation is that dose that will produce as many new mutations within one generation as those arising spontaneously. Until recently, the doubling dose derived from mouse studies alone was estimated to be 1 Gy, and this was applied to estimate hereditary effects in human populations receiving low-dose exposures over many generations.

39. In 2001 the Committee revised its methods to:

(a) Include new estimates of the spontaneous mutation rate in humans;

(b) Allow for the lethal effects of some mutations on embryo development and for others that will disappear from the population because they prevent or greatly reduce the likelihood of human reproduction;

(c) Use a revised relationship between the input of new mutations into the population and the incidence of heritable disease.

40. In the light of the above revisions and other analyses, the Committee was able to provide not only a new estimate of the risk of single-gene dominantly inherited diseases, but also for the first time an estimate of the risk of multifactorial diseases. These estimates are shown in table 2 and are based on a scientifically more robust framework than was available prior to 2001. Rapid advances in DNA-sequencing technology and understanding of human genetic variation may provide more direct information on heritable risk in the future.

## C. Radiation-associated non-cancer diseases

41. Radiation exposure of the developing embryo or foetus during pregnancy can also contribute to the appearance of non-cancer diseases in children. In addition to the induction of congenital malformations, the central nervous system is particularly affected. Two main factors influence risk: the radiation dose and the specific stage of development of the embryo or foetus at the time of exposure. Mainly on the basis of animal studies and some observations following high-dose exposures of pregnant women, the Committee considers that there is a threshold for these effects at about 100 mGy.

42. Evidence has accumulated that the risk of common diseases in addition to that of cancer can increase following irradiation, at least at moderate to high doses. The main source of this evidence is epidemiological studies of data from the survivors of the atomic bombings in Japan, particularly on circulatory disease. In its 2006 report,[10] the Committee reviewed evidence both from the studies of the survivors of the atomic bombings and from a range of investigations of other irradiated groups. The difficulties encountered in undertaking this analysis included: the high background occurrence of these diseases in non-irradiated populations; making adequate allowance for factors other than radiation exposure (e.g. smoking, cholesterol levels, inherited predisposition); and a lack of identified cellular mechanisms involved in their development. The only clear evidence for an excess risk of fatal cardiovascular disease associated with radiation exposure, at doses to the heart below about 1 to 2 Gy, comes from the data on the atomic bombing survivors. The other studies reviewed by the Committee showed evidence for excess cardiovascular disease at higher doses. For all other non-cancer diseases combined, the same general conclusion reached for cardiovascular disease applies. The Committee's review was not able to draw any conclusions about a direct causal relationship between irradiation at doses below about 1 to 2 Gy and excess incidence of cardiovascular and other non-cancer diseases. The shape of the dose-response relationship at low doses for these diseases is not yet clear.

43. There is emerging evidence from recent epidemiological studies indicating elevated risks of non-cancer diseases below doses of 1 to 2 Gy, and in some cases much lower. However, the associated mechanisms are still unclear and the estimation of risks at low doses remains problematic. This is an area of active research, and the Committee will continue to keep developments under review.

44. In 2006 the Committee also provided an assessment of the effects of ionizing radiation on the immune system.[11] In principle, if radiation served to enhance or diminish the capacity of the body to mount an immune response to an infection, cancer or other disease, the risk of any disease due to radiation exposure could be affected. While many studies were examined, it remained impossible to make a clear judgement as to whether the effects on the immune system of radiation at low doses served to stimulate or suppress immune responses.

45. Finally, the Committee notes that recent studies also suggest that an increased incidence of cataracts may be associated with low-dose radiation exposure. The induction of such abnormalities in the lens of the eye has been recognized for some years as an effect of high-dose exposures. As with circulatory diseases, the Committee will continue to monitor and review new findings in this area. Furthermore, mechanisms of potential relevance to explaining radiation-induced disease, such as genomic instability and bystander effects, will be kept under review, along with emerging concepts and technologies that might contribute to a more complete understanding of the health effects of low-dose radiation exposure and of the mechanisms that explain these effects.